Modern Concepts of Cardiovascular Disease

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EPIDEMIOLOGY AND CARDIOVASCULAR DISEASE OF MIDDLE AGE: PART I*†

Medical knowledge may be acquired in three principal ways: through clinical observation, laboratory experiment, and population study or epidemiology. Each method has its own contribution to make; each is the right way to ask some questions, but is the wrong way to ask others. In this paper, I will illustrate some uses of epidemiology in learning about coronary disease and hypertension. I will not try to summarise the numerous investigations now under way in different parts of the world and will present findings only to illustrate argument and principle. Illustrations are often drawn from the work of the Social Medicine Research Unit.

Rise of Ischaemic Heart Disease

Sigerist made the statement that every civilisation produces its own diseases. Is it a fact that ischaemic heart disease (IHD) has been increasing during the present century? Figure 1 is an example of the most elementary kind of epidemiology; it gives the death rate from all causes in the population of England and Wales[50] during the past 100 years, the number of deaths/persons in the relevant ages at various periods giving the simple rates that are plotted. The figure provides a background for the perennial discussion as to whether there is more "coronary thrombosis" now than there used to be. Mortality among middle-aged men has been stationary at about 22 per 1,000 since the end of the recent war. The male death rate a hundred years ago was 10 per cent higher than the female; now it is 100 per cent higher. Examination of the death rates from particular causes shows that men as well as women have benefited from the recent advances in the treatment of infections and other conditions, so something else must have happened to wipe out these benefits. The suggestion from individual clinical experience (not a good way of answering this question) and national cause-of-death statistics (the right way, but carrying an enormous technical error) is that an increase of ischaemic heart disease is the principal factor in this recent, very unsatisfactory trend of mortality in middle-aged men. There are other bits of information: in two segments of the population, British physicians and London busmen, observed in detail by the author and his colleagues since the late 1940's, IHD has been increasing. Study of the medico-legal necropsies for the frequency of ruptured ventricle—a very simple indicator of IHD—shows a similar increase over the years.

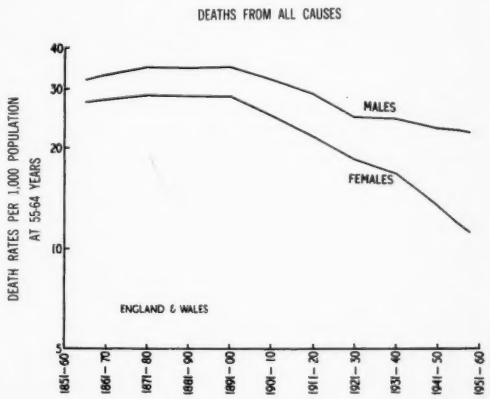

Figure 1.

* From the Social Medicine Research Unit of the Medical Research Council, London Hospital, London, England.

† Part II of this article will appear in the January, 1961, issue.

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Table 1

Recent History of Coronary "Atherosclerosis": Prevalence of Coronary Artery Disease at Post-mortem in Deaths from Other Causes than Ischaemic Heart Disease in Men

Condition of coronary arteries	Deaths from accidents, infections, cancer, etc.; ages 45-54		Deaths from hypertension, vascular disease, diabetes, etc.; ages 45-64	
	1908-13*	1954-56†	1908-13*	1954-56†
	%	%	%	%
Moderate and extensive mural atheroma	51	30	70	60
Calcification present	17	6.7	32	23
Narrowing of lumen present	3.8	11	12	28
Severe narrowing of lumen	1.3	4.8	5.8	14
Ischaemic myocardial fibrosis	% 0.8	% 3.8	% 5.3	% 16
Number of necropsies	373	821	171	728

* London Hospital necropsy reports.

† National necropsy survey, Great Britain (results are very similar if modern London Hospital data are used instead).

(Adapted from Morris, 1951;[35] Morris and Crawford, 1958.[37])

Is it possible to be more specific about the pathology of the increase? Table 1 is the opposite of figure 1 in terms of "hardness" of the evidence, because post-mortem data contains an unknown number of sources of error and it is unlikely that any one study can deal with even the recognised sources; also, because the issue of observer bias–"believing is seeing" or "seeing what is being looked for"–arises in extreme form. The table raises the possibility that atheroma of the walls of the coronary arteries has not increased during this century. (Indeed, there was so much atheroma before the first World War, it is difficult to imagine that it could have increased.) But severe obstruction of the coronary lumen, such as coronary thrombosis as a cause of death (in every adequate hospital series known to the author), and severe stenosis, actual occlusion, and fibrous obliteration, the result mostly of organised thrombosis, in men dying from other causes appears to be commoner. The hypothesis to bear in mind from the historical material, to guide further study and in particular the search for environmental factors, is that there has been an increase of coronary thrombosis. This, it is suggested, is the "modern epidemic." To my mind, it is impossible to make useful estimates of the size of the increase.

Recent History of Hypertension. The other important question, whether there is more essential hypertension per head of middle-aged population than, say, 50 years ago, is even more difficult to answer. The death rate from cerebrovascular disease has not increased in our country, nor has the frequency at necropsy of left ventricular hypertrophy in the absence of valvular disease. How much hypertension is involved in the increase of IHD cannot be guessed. We can only say that the present evidence is very sketchy; what little there is does not indicate any dramatic change in the frequency of essential hypertension. Long-term community surveys are needed and have already been started.[12, 14, 31]

Community Picture

Epidemiology is concerned with disease as a public health problem: how much there is of it in the population and how this is distributed by

Table 2

Estimates of Prevalence of Coronary Disease in Men Aged 60-64: Great Britain, late 1950's

Almost 100% have atheroma of the walls of the coronary arteries.

About 25% have extensive, confluent coronary atheroma.

About 33% have visible narrowing of a main coronary artery.

About 15% have occlusion of a main coronary artery.

Perhaps 5% have acute major thrombosis of a main artery, p.a.

About 15 or 20% have ischaemic myocardial pathology.

About 10% have clinical ischaemic heart disease or a history of it.

About 5% are ill with clinical disease p.a.

About 2% have clinical disease for the first time p.a. (incidence).

Between ½ and 1% die of IHD p.a.

(Adapted from Morris, 1957.[36]) Of these figures, the one most likely to be very wrong is the 10% for clinical disease.[12, 14, 53]

sex and age in different places and among the main social groups. Table 2 makes some rough estimates, although they are probably accurate enough for order of magnitude of coronary disease in Britain in the late 1950's. These are estimates of the average; there will be a wide range on either side to include variation among social and other groups. The table imagines that men in their early sixties are observed over a single year and describes events in it. My guess is that a corresponding American table would show even worse pathology.

Table 3

Morbidity from Cardiovascular Disease in General Practice During One Year: Patients Consulting per 1,000 on Practitioners' Lists, Ages 45-64

Diagnosis	Men	Women
Coronary disease	17	7.4
Hypertension and hypertensive heart disease	15	37
Vascular disease of nervous system	4.3	3.7
Peripheral arterial disease; other arteriosclerosis, etc.	5.4	3.8
Rheumatic fever and heart disease	1.6	3.3
Varicose veins, phlebitis, etc.	14	33
Haemorrhoids	12	8.8
Diabetes	4.7	6.5
Bronchitis; emphysema	90	64
Total patients consulting per 1,000 on lists; all diagnoses	604	667

Great Britain: 106 general practices containing 383,000 people, of whom 100,000 were aged 45-64. (Adapted from Logan and Cushion, 1960,[27] by permission of the Controller of Her Britannic Majesty's Stationery Office.)

Table 3, drawn from a recent large-scale study of morbidity in general practice in our country, shows the various types of cardiovascular disease, in their proportions, as they turn up in a segment of the population of about 400,000 ordinary people. Comparison of the size of these rates is interesting. The level, or "plane," of diagnosis in many cases will be, of course, that current in general practice. The author would prefer to have given figures for pulmonary heart disease rather than merely for "bronchitis and emphysema," but has been unable to find the necessary data. A survey in general practice, recording every case with signs of congestive failure and its presumed aetiology, would provide data for a large-scale map that could be usefully set alongside similar information about IHD, etc. There is no doubt that cor pulmonale is a major cause of heart failure and death in our country, apparently commoner here than in the U.S.A. or in Scandinavia. Why this is so is now being investigated in comparative international studies.

Medical Care. Estimates of the frequency of disease in populations, or population groups, have various uses. They provide a baseline for the assessment and planning of health services. Thus, simple sums suggest that only a small fraction, in fact it may be less than one-tenth, of the clinical "coronaries" of table 2 are admitted to hospital. The challenge that the middle-aged cardiac presents for rehabilitation service is also indicated, although figures for men in their fifties are more revealing. I might hazard the estimate that there are 50,000 men in Britain, aged 50-54, with clinical IHD, who possibly need rehabilitation. Better data with far more detail than in tables 2 and 3 are badly needed, and their collection, particularly from clinical material, would make a useful and professionally very worthwhile enterprise in any sizeable community.

Table 2 has other functions. It relates clinical disease, the tip of the iceberg, to subclinical, occult or latent disease. It indicates such things as the scope for case-finding–between the 10 and the 15 or 20 per cent groups. The frequency of subclinical disease demonstrates the difficulty of finding satisfactory "controls" for studies of coronary patients.

To turn to the distribution of disease in social groups: the classic observation is that mortality from IHD differs in the social classes of England and Wales. There is quite a clear fall from social class I (leading professions) through the lesser professions (II) and skilled workers (III) to social class IV (the partly skilled workers).[26] Such a study of the social variation of disease is often the beginning of a search for causes, of formulating hypotheses about *why*, and *what* in the composition and way of life of these groups might be responsible. Thus, it was because of such findings as those on social class, together with some hunches from clinical experience, that the author and his colleagues began, in London, an intensive study of occupation in relation to IHD. However, before proceeding to this, the chief function of epidemiology, there is another that must be considered.

Completing the Clinical Picture

Any clinician's experience with the chronic diseases must be incomplete. Ischaemic heart disease, for instance, presents itself differently in general practice, consulting practice, industrial practice, in hospital out-patients, "casualty" departments, medical wards, surgical wards, to the coroner's pathologist or the general pathologist. Moreover, all clinical experience is likely to be incomplete. Mild cases, and cases that are by no means mild, may not attract clinical attention for one reason or another, and there is no assurance, even in very large series, that various manifesta-

tions are included in their proper proportion. The incomplete clinical picture may thus be an incorrect one.[36] Epidemiological surveys, by attempting a total account of a disease as it occurs in a defined population and not being restricted to any particular set of patients, may assist in completing and correcting the clinical picture. The extent to which this can be done will depend on how far the problems of epidemiological method can be resolved and rates produced that truly reflect what is happening in the population.

Hospital and Community. When a ruptured ventricle from IHD was investigated as a possible indicator of the frequency of that disease, it was found to be a rather uncommon condition. In all of London's hospitals, including the 13 main teaching hospitals, there were 80 or 85 cases during the two years 1957-58. This was illusory, however, and the reason was not hard to find; in London, most of these cases are dealt with by the coroner's pathologist (medical examiner) in the mortuary of the Coroner's Court. Thus, in a 65 per cent sample of coroners' pathology during the same years, and from the same segment of the population, there were 345 cases. Scaling up these figures to produce an estimate of the total incidence (this is the correct term) for the 3¼ million people of London, suggests that ruptured ventricle is by no means a rare condition (table 4). It may be said that there are about 300 cases per year, or 100 per million of the population.

Table 4

Hospital and Community: Ruptured Ventricle in London During 1957 and 1958; Number of Cases Found at Necropsy

Age	13 main teaching hospitals		Other hospitals*		Coroners' mortuaries†		Totals	
	M.	F.	M.	F.	M.	F.	M.	F.
Under 70	15	4	13	4	86	60	114	68
70 and over	4	13	11	19	145	240	160	272

* Adjusted from > 95% sample.
† Scaled up from 65% sample.
(Adapted from Crawford and Morris, 1960.[10])

Why were there so few in hospitals? The answer lies in a feature of ruptured ventricle that has emerged clearly from the coroners' cases. It is a disease especially of elderly people, and there are special difficulties in their admission to hospital. Moreover, the histories are often peculiarly slight, probably because of age, so that if these cases were taken into hospital they would be classified as "B(rought) I(n) D(ead)"; for these cases, however, other arrangements are made in London through the system of coroners. The largest number was in elderly ladies. But this does not mean that the actual rate of occurrence is greatest in them because there are in London over 1½ times as many females over 70 as males, due in some part to the far higher death rate of males during middle age from IHD. The great number of cases in elderly ladies probably reflects something of an equal incidence in the two sexes among the old. This recalls a clinical hunch about the natural history of IHD, which so far has been difficult to prove.

Clinical Medicine and Epidemiology. The principle is this: Clinicians deal with patients, epidemiologists with populations. In studying the frequency of disease, the epidemiologist, for the numerator of the rate he is always trying to produce, must find all the cases possessing the defined characteristics in the population (the denominator), whether they are "patients" or not. Usually this requires special surveys, and it is on these that the epidemiologist spends most of his time. In the instance used, the survey was simple because of the administrative arrangements in London whereby almost every case of unexplained death, including ruptured hearts, routinely undergoes necropsy anyhow. Mere addition of hospital numbers and those from the coroners' mortuaries gives an adequate population picture, although even this may be an underestimate from cases wrongly diagnosed before death and not brought to necropsy. The position would be quite different in a city like New York where, I am told, only a small fraction of the cases of unexplained death, which are manifestly due to natural causes, undergoes post-mortem examination.

To sum up: the clinical picture in London of ruptured ventricle and, in particular, in the teaching hospitals, which are the source of most statements on pathology, is incorrect, particularly as to sex, age, and the role played by anticoagulants. The latter were involved in 16 per cent of the hospital cases, but in only 2 per cent of the total.

Irregular Epidemiology. This happy phrase, it has been reported, was coined at a recent World Health Organization (WHO) meeting in a plea for the amateur epidemiologist and for the encouragement of the clinician to extend his analysis and inferences to the section of the population from which his patients are drawn. No one with even the sketchiest history of epidemiology can be unenthusiastic about such a plea or would advocate anything like a professional "closed shop" (I hope this has the same meaning on both sides of the Atlantic). But the foregoing discussion may be read as an indicator of the kind of consideration to be borne in mind when translating hospital numbers and proportions into generalisations about what is happening in communities, and it is these that are involved in state-

ments such as that a disease is commoner in one race, or sex, or age than another.

Search for Causes of Ischaemic Heart Disease

A case of ischaemic heart disease or the occurrence of IHD in a population, like any historical event, has many causes. At present, epidemiological methods are being used to seek out the causes by comparison of the coronary experience of groups of men with varying environments and ways of living, such as different diets or jobs, or personal characteristics, e.g., family history or physique. Postulated causes are being related, both to final IHD and to intermediate causes or mechanisms, such as hypercholesterolaemia and hypertension.

Peoples and Places. Year after year, substantial differences between countries in mortality from IHD are reported by the World Health Organization. Even when comparisons are restricted to highly developed countries with comparable levels of medical practice and procedures of death certification, the differences remain substantial. Thus, in Western Europe, the death rate from IHD in Finland is almost three times that of Belgium, while U.S. whites, it will be recalled, have the highest coronary death rates of all reported to WHO. Perhaps the finding which sheds the most light on the aetiology of IHD is that underdeveloped populations have little of it compared with those which are highly developed. The main inference drawn from these various geographical differences is that diet is largely responsible; this has been a tremendous stimulus to the laboratory study of fats and atheroma.

Brock and his colleagues have made use of the "natural experiment" or "experiment of opportunity" provided by the existence in Cape Town, side by side, of three racial groups who are also at different stages of social and economic development. Bronte-Stewart has kindly assisted the author in compiling table 5, summarising the main results of these ongoing studies (personal communication, 1960). Clinical and laboratory sample investigations confirm, amplify, and to some extent may explain the gross contrasts found in IHD. But the last two columns illustrate starkly the difficulties of meaningful comparison and the danger of applying these findings to other social and biological situations.

Keys, with colleagues in many parts of the world,[7, 25] has been engaged in discovering such contrasts and establishing the facts of IHD in populations with different diets. In the chronic diseases, in particular, it is crucial to test hypotheses derived from laboratory experiment (that on saturated fat, for example) in actual human experience. In exploration of one natural experiment after another, Keys seeks progressively to

Table 5

Cape Town Interracial Studies of IHD and Its Aetiology: Middle-aged Men

Population	Mortality from IHD per 100,000*	ECG. of IHD† %	Coronary atherosclerosis‡ Extensive mural atheroma %	Severe lumen narrowing %	Casual diastolic blood pressure over 100 mm. Hg %	Blood cholesterol Total mg. %	Blood cholesterol β-lipoprotein %	Blood coagulation§ Factor VII % (Serum)	Blood coagulation§ AHG %	Skinfold‖ 14 mm. and + %	Diet** Animal fat Gm.	Diet** % Cal. from fat	ESR over 8 %	Infant†† mortality
European	628	13	20	14	8	242	83	92	120	47	82	40	3.5	21
Cape coloured	274	6.2	16	9.7	12	195	77	—	—	21	60	25	15	77
Bantu	very low	0	5.6	0	8.3	168	72	75	163	27	40	17	44	190

* Age 55-59, 1955. Other values are averages for sample population surveys as indicated and refer to varying age-matched ranges within 40-59 years.
† Miscellaneous out-patient ECG.'s, 40-49.
‡ Deaths from accidents, infections, cancer, etc., 40-59.
§ Wider age range and rather younger.
‖ 40-54 years. Triceps.
** Semiquantitative.
†† 1954-55 (corrected). Occupational distribution: Europeans (professional, business, white-collar); Bantu (labourers); Cape coloured (intermediate, but much nearer Bantu).
(Bronte-Stewart, B., and Brock, J. F., et al., personal communications, 1956; 3, 3, 29, 44, 46, 47)

Table 6

Two Populations in Yugoslavia: Studies of IHD and Its Aetiology in Men Aged 40-59

Population	ECG. abnormality S-T depression %	ECG. abnormality T-ve %	Diastolic blood pressure mm. Hg	Serum cholesterol mg. %	Skinfolds triceps and subscapular mm.	Diet—% Cals.* From fats	Diet—% Cals.* From saturated fatty acids
Slavonia	8.9	7.0	81	197	14	32	13
Dalmatia	3.8	1.8	82	185	14	31	6.7

* By ad hoc local chemical analysis. Almost 100% of the 1,300-1,400 men eligible were examined. Figures are averages. (Keys, A., Anderson, J. T., Brozek, J., Buzina, R., Ferber, E., Fidanza, F., Mohacek, I., Punsar, S., Rautaharju, P., Simonson, E., and Tiefenback, B., personal communication, 1960.)

improve the diagnosis of IHD and to "control" the dietary factor.

Table 6, from a recent expedition to Yugoslavia, illustrates the present stage of his enquiry; these are two populations engaged in very similar jobs. Tables 5 and 6 represent major advances in the indirect studies of international death rates in relation to food availability.*

Results are now beginning to come from Evans County (Hames, C. G., and Greenberg, B. G., personal communication, 1960). This epidemiological investigation, based on district general practice (an "ordinary" population) and using cardiology and basic sciences, is focused on the excess of IHD reported in the death statistics of U.S. white men compared with Negroes. Current comparisons are being supplemented by studies seeking out the beginnings of the disease. Already interesting differences are being found among children: the β-lipoprotein fraction of the blood cholesterol is consistently higher in white boys and girls than in coloured. This study would have delighted Sir James Mackenzie.

Heredity and Environment. An indirect approach to the elucidation of the environmental component in IHD uses geographical pathology and compares the same racial groups in different environments. There is growing evidence, in particular from Israel, that with adoption of Western ways of life blood lipid levels and the rate of IHD increase.[7, 15]

Hypothesis on Exercise

Figure 2 illustrates the statement and testing of a hypothesis in our unit by epidemiological methods.[23, 34, 36, 37, 38] When government clerks were found to have the same kind of excess of IHD over postmen as bus drivers had over conductors (section 1), suspicion hardened that differences in the habitual physical activity of these jobs were involved in the difference of IHD. A hypothesis was formulated to guide further investigation. Characteristic of "causes" of IHD that are now being postulated, the protection afforded by habitual physical activity is seen most clearly among the younger men in early middle age (compare hypercholesterolaemia and, in our country, smoking). There is a chance then, it may be suggested, for the specific cause to be especially effective, and thus to be detected more readily before the multiplication of widespread pathogenic factors obscures the picture in later middle age. Several points in these investigations may be noticed. Occupational groups were selected that were large, homogeneous (all drivers, for example, drive for most of their working day), and of similar social economic status (there is less than a dollar a week difference in pay between drivers and conductors). Multiple, independent attacks were made on the problem (sections 1, 2, and 7) in the hope that the same kinds of bias and fallacy would not always be turning up. Various stages of the disease were investigated, from the incidence of the first clinical episode (section 1) to final mortality (section 7); also various types from "sudden death" to focal myocardial fibrosis. Different "levels" of epidemiological study are illustrated: national vital statistics, e.g., the death rates by social class; large-scale morbidity study (the incidence rates in section 1, for example), and small-scale clinical sample surveys, e.g., the figures on blood pressure, blood lipids, subcutaneous fat (sections 4b, 5, 6b). A wide range of measurements had thus to be employed.

Pathogenesis. Mechanisms were sought in the distribution of coronary artery disease, in blood pressure and blood lipid levels. That different men select different jobs is axiomatic. A tentative approach was made to constitution and physique by using data easily available on uniform size. The men chosen for illustration in section 6a, by the way, are all 5′8″ or 5′9″ tall. The validity of this crude measure of fatness was established by more accurate clinical measurement of samples of the men. So far, the indication from these studies is that occupational

* Enough has been written on the use of international statistics.[32, 33, 56]

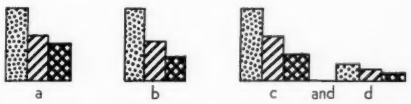
1. Incidence of Ischaemic Heart Disease

a) In London busmen
(drivers and conductors)
(1) All forms, age 35-65
(2) Sudden death 35-50

b) In government employees
(clerks and postmen)
(1) All forms, 35-60
(2) Rapidly fatal infarction 35-50

2. Ischaemic Myocardial Fibrosis

Deaths from other than ischaemic heart disease 45-70
a) All forms b) Large healed infarcts c) Focal myocardial fibrosis in cases with essential hypertension d) in cases without hypertension



3. Coronary Artery Disease (Necropsy Findings)

Deaths from other than ischaemic heart disease 45-70
a) Extensive atheroma of walls b) Calcification present
c) Slight narrowing of lumen d) Lumen occlusion

4. Essential Hypertension

a) Evidence as found at necropsy
(1) E. H. related to death
(2) E. H. incidental
(3) No hypertension

b) Casual diastolic blood pressure readings
Over 105 mm. Hg drivers and conductors

Age	Driver	Conductor
	Per cent	
40-49	4.2	0
50-59	14.1	6.2
60-64	13.5	11.1

5. Blood Lipids

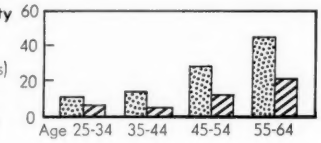
London busmen
(drivers and conductors)
50-59

Lipids	Driver	Conductor
	mg. %	
Plasma Cholesterol	254	238
β-lipoprotein	199	191
Sf 0-20	403	354
Sf 20-400	176	89

(Courtauld Institute of Biochemistry, Middlesex Hospital)

6. Physique and Obesity

In London busmen
(drivers and conductors)
a) % Uniform trouser waist 38" and more



b) Fatness
Skinfold Thickness
Triceps, subscapular, suprailiac

c) Sudden death from ischaemic heart disease Age 35-50, 1949-58

Trouser waist	Driver	Conductor
	Rates per 1,000	
32" and less	0.8	0.2
34"-37"	0.5	0.2
38" and more	1.0	–

7. Ischaemic Heart Disease: Physical Activity of Work and Social Class*

a) Mortality, England and Wales

Age 45-54, 1949-53

Social Class	Light	Active	Heavy
	Rates per 100,000		
I	176	–	–
II	167	118	–
III	173	137	111
IV	153	121	116
V	175	145	91
All	173	135	102

b) Ischaemic myocardial fibrosis in deaths from other than ischaemic heart disease

Age 45-70

Social Class	Light	Active	Heavy
	Per cent		
I	13.7	–	–
II	13.4	9.2	–
III	13.8	8.0	8.2
IV	13.5	10.1	4.9
V	8.9	9.0	7.1
All	13.4	8.7	6.8

*Social Class I (leading professions) to V (unskilled workers).

Figure 2. Physical Activity and Coronary Disease: An Epidemiological Hypothesis and its Testing

differences in obesity are brought by the men with them into their jobs but are not responsible for the difference found in IHD (section 6c). Measurements are being taken in the clinical survey to give a somatotype of each man.

It must be noticed that all these observations are on occupation; this kind of study is unlikely to give information on habitual or sudden overexertion. Moreover, no data are available so far on the value, if any, of exercise in leisure, which is the implication for preventive medicine of such observations as are found in figure 2 (although I have been told that the Russians, with their different approach, are instituting mass physical culture in the factory). Finally, it is evident that physically active workers still have plenty of IHD–in fact, far too much. If this is a cause, evidently there are greater ones. This cause, too, may be effective only in some situations;[1, 34] thus, some studies in the U.S.A. have found no relationship.[15, 16]

* * *

The main importance of the exercise factor may be its contribution to the *recent increase* of coronary thrombosis and IHD. There has been a drastic reduction of physical effort in work, transportation, and leisure from recent technological changes. This is likely to have affected men more than women; perhaps it is one of the social changes underlying the biological changes of figure 1. Other epochal changes in the "epidemic constitution"[18] of the time have been cigarette smoking, also sex-linked in our country, and the use of margarine. The former has a rather complicated,[13] the latter no obvious, relationship with IHD. The nature of nervous strain may be very different; it could be that personality structure has also changed.*

References

All references for Parts I and II of this article will appear at the end of Part II in the January, 1961, issue.

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* Recent psychological-social studies, of Wolf,[55] Russek and Zohman,[45] and Friedman and Rosenman,[17] for example, will not be considered. They are of particular interest to social medicine because they relate the occurrence, or increase, of IHD to current pictures of character and neurosis (by Erikson and Riesman, e.g.) and thus, in turn, to more profound changes in social life that have not been discussed here. Such are: the speed of change, growth in scale of organisation, mass communication, the confusion of roles, the concern with status. (America today and, it seems, Europe tomorrow.) Meanwhile, there is no epidemiological base to the published studies, no estimate of the frequency of the personality described in any population or in representative series of patients with IHD. Moreover, it may well be that the picture presented of the "coronary" patient is not, in fact, at all specific. This would not render it less interesting or less important.

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